**WHAT IS CLAIMED IS:**

**1.** A method for identifying a therapeutic agent for use in treating a CAR-mediated disorder or condition, the method comprising:

identifying a candidate therapeutic agent by screening one or more compounds to determine whether said compounds can modulate a CAR-mediated intermolecular interaction;

administering the candidate therapeutic agent to a test mammal; and

determining whether the level of a cholesterol indicator is modulated in said test mammal.

**2.** The method of claim **1**, wherein said candidate therapeutic agent is 5β-pregnan-3,20-dione.

**3.** The method of claim **1**, wherein said CAR-mediated disorder or condition is selected from the group consisting of: hypercholesterolemia, lipid disorders, atherosclerosis, and cardiovascular disorders.

**4.** The method of claim **1**, wherein the mammal is a cholesterol-elevated mammal.

**5.** The method of claim **4**, wherein the test mammal has a disruption in both CAR alleles.

**6.** The method of claim **1,** wherein said cholesterol indicator is the level of serum cholesterol.

**7.** The method of claim **1**, wherein said cholesterol indicator is the level of a member selected from the group consisting of HDL cholesterol, LDL cholesterol, and VLDL cholesterol.

**8.** The method of claim **1**, wherein said cholesterol indicator is the mRNA level of a gene involved in the regulation of cholesterol levels.

**9.** The method of claim **1**, wherein said CAR-mediated intermolecular interaction is CAR-mediated gene expression.

**10.** The method of claim **9**, wherein the ability of said candidate therapeutic agent to modulate CAR-mediated gene expression is assessed by:

providing a cell that comprises:

a) a polynucleotide encoding a fusion polypeptide that comprises: 1) an amino acid sequence that comprises a DNA binding domain of a polypeptide; and 2) a ligand binding domain that is substantially identical to a ligand binding domain of CAR; and

b) a reporter gene construct which comprises a response element to which the DNA binding domain can bind, wherein the response element is operably linked to a promoter that is operative in the cell and the promoter is operably linked to a reporter gene; and

contacting said cell with said candidate therapeutic agent; and

determining whether said reporter gene is expressed at a higher or lower level in the presence of said candidate therapeutic agent as compared to expression in the absence of said candidate therapeutic agent.

**11.** The method of claim **10**, wherein said candidate therapeutic agent is 5β-pregnan-3,20-dione.

**12.** The method of claim **10**, wherein said DNA binding domain is substantially identical to a DNA binding domain from a polypeptide selected from the group consisting of: CAR, a GAL4 transcription factor, an estrogen receptor, a progesterone receptor, a glucocorticoid receptor, an androgen receptor, a mineralcorticoid receptor, a vitamin D receptor, a retinoid receptor, and a thyroid hormone receptor.

**13.** The method of claim **12**, wherein said DNA binding domain is a CAR DNA binding domain and the response element is a CAR response element.

**14.** The method of claim **13**, wherein said CAR response element is a DR-5 element or a DR-4 element.

**15.** The method of claim **10**, wherein said reporter gene encodes a marker protein selected from the group consisting of: luciferase, alkaline phosphatase, beta-galactosidase, chloramphenicol acetyltransferase and green fluorescent protein.

**16.** The method of claim **1**, wherein said CAR-mediated intermolecular interaction is the binding of a polypeptide that comprises a CAR ligand binding domain to a ligand for CAR.

**17.** The method of claim **16**, wherein said polypeptide is a CARα or a CARβ.

**18.** The method of claim **16**, wherein said ligand for CAR comprises a sensor peptide.

**19.** The method of claim **18**, wherein said ligand for CAR comprises a receptor binding domain of a coactivator or a corepressor.

**20.** The method of claim **19**, wherein said coactivator is SRC-1.

**21.** The method of claim **20**, wherein said sensor peptide is rhodamine labeled ILRKLLQE.

**22.** The method of claim **16**, wherein the binding of the polypeptide that comprises a CAR ligand binding domain to the ligand for CAR is determined in the presence of a naturally occurring ligand for CAR.

**23.** The method of claim **22**, wherein said naturally occurring ligand for CAR is 5β-pregnan-3,20-dione.

**24.** The method of claim **16**, wherein said method comprises determining whether said compound can inhibit the interaction between the CAR ligand binding domain and the CAR ligand.

**25.** The method of claim **24**, wherein said CAR ligand is labeled.

**26.** The method of claim **25**, wherein said CAR ligand is radiolabeled.

**27.** The method of claim **24**, wherein said CAR ligand is labeled with a fluorophore.

**28.** The method of claim **27**, wherein said method comprises a fluorescence polarization assay.

**29.** The method of claim **27**, wherein said method comprises a fluorescence resonance energy transfer assay.

**30.** The method of claim **27**, wherein said CAR is labeled with a fluorophore.

**31.** The method of claim **30**, wherein said method comprises a fluorescence resonance energy transfer assay or a fluorescence polarization assay.

**32.** The method of claim **24**, wherein said CAR ligand is selected from the group consisting of:

5α-androst-16-en-3α-ol, 5α-androst-16-en-3α-ol acetate, 5α-androstane-3α-ol, 5α-androst-16-en-3α-ol acetate and 5β-pregnan-3,20-dione.

**33.** A method for identifying a therapeutic agent for use in treating a CAR-mediated disorder or condition the method comprising:

administering a compound to a CAR compromised mammal; and

determining whether administration of the compound results in a change in cholesterol level compared to a mammal to which the compound is not administered.

**34.** The method of claim **33**, wherein the method further comprises administering the compound to a CAR non-compromised mammal and comparing the effect on the cholesterol level indicator of administering the compound to that of administering the compound to the CAR compromised mammal.

**35.** The method of claim **33**, wherein said cholesterol level indicator is the level of serum cholesterol.

**36.** The method of claim **33**, wherein said cholesterol level indicator is the level of a member selected from the group consisting of HDL cholesterol, LDL cholesterol, and VLDL cholesterol.

**37.** The method of claim **33**, wherein said cholesterol level indicator is the mRNA level of a gene involved in the regulation of cholesterol levels.

**38.** The method of claim **33**, wherein said CAR compromised mammal is a mammal having a disruption in both CAR alleles.

**39.** The method of claim **38**, wherein said CAR compromised mammal is a mouse.

**40.** The method of claim **38**, wherein said disruption occurs in the coding region for the DNA binding domain of CAR.

**41.** The method of claim **38**, wherein said disruption in a CAR allele comprises an insertion at codons for amino acid positions from about amino acid 21 to about amino acid 86 of CARβ.

**42.** A method for treating a CAR-mediated disorder or condition, the method comprising:

administering to a mammal having a CAR-mediated disorder or condition an effective amount of a therapeutic agent that modulates CAR-mediated regulation of cholesterol levels.

**43.** The method of claim **42**, wherein said therapeutic agent is identified by:

screening one or more compounds to determine whether said compounds can modulate a CAR-mediated intermolecular interaction;

administering the candidate therapeutic agent to a test mammal; and

determining whether the level of a cholesterol indicator is affected in said test mammal.

**44.** The method of claim **42**, wherein said CAR-mediated disorder or condition is selected from the group consisting of: hypercholesterolemia, lipid disorders, atherosclerosis, and cardiovascular disorders.

**45.** A non-human mammal having a genome that comprises a disruption in at least one CAR allele.

**46.** The non-human mammal of claim **45**, wherein said disruption comprises an insertion, deletion or mutation in a region of the CAR allele that encodes for a DNA binding domain of CAR.

**47.** The non-human mammal of claim **46**, wherein said disruption comprises an insertion at codons for amino acid positions from 21 to about 86 of CARβ.

**48.** A non-human mammal having a genome that comprises a disruption in both CAR alleles.

**49.** The non-human mammal of claim **48**, wherein said disruption comprises an insertion, deletion or mutation in a region of the CAR allele that encodes for a DNA binding domain of CAR.

**50.** The non-human mammal of claim **48**, wherein said disruption comprises an insertion at codons for amino acid positions from 21 to about 86 of CARβ.

**51.** The non-human mammal of claim **48**, wherein said non-human mammal exhibits an increased level of serum cholesterol relative to a wild-type mammal.

**52.** A method for producing a transgenic non-human mammal having a genome that comprises a disrupted CAR allele, the method comprising:

introducing into embryonic stem cells a polynucleotide that comprises a coding region for a portion of a CAR polypeptide, wherein the polynucleotide sequence includes a disruption in the coding region of a portion of said CAR polypeptide;

identifying a cell into which said polynucleotide sequence has been integrated into an endogenous CAR allele;

introducing said cell into a blastocyst, thereby forming a transgenic blastocyst;

implanting said transgenic blastocyst into a pseudopregnant mammal and allowing said pseudopregnant mammal give birth to a transgenic mammal.

**53.** The method of claim **52**, wherein said transgenic mammal contains a disrupted CAR allele in its germline.

**54.** The method of claim **53**, further comprising breeding said transgenic mammal to generate a heterozygous mammal comprising a disrupted CAR allele.

**55.** The method of claim **53**, further comprising mating a male and a female mammal each heterozygous for said disrupted CAR allele, to form progeny that are homozygous for said disrupted CAR allele.

**56.** The method of claim **52**, wherein said disrupted CAR allele comprises an insertion into a region of the CAR allele that codes for a DNA binding domain of CAR.

**57.** The method of claim **52**, wherein said disrupted CAR allele comprises an insertion at codons for amino acid positions from about 21 to about 86 of CARβ.

**58.** The method of claim **56**, wherein said insertion comprises a selectable marker gene.

**59.** The method of claim **58**, wherein said marker gene encodes for neomycin resistance.